THESIS.

CHRONIC PULMONARY INDURATIONS.

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During the last 80 years the subject of pulmonary indurations has from time to time been discussed by various medical writers, indeed one cannot look through any monograph, or text book dealing with lung diseases, from 1810 to present date, without finding some mention and expression of opinion as to the causation, the course, and results of chronic induration of the lung. But although this subject has been so frequently discussed, yet no really definite conclusion has been arrived at. Instead of all views being brought into line, and deductions drawn therefrom we have still, when dealing with this subject, a multiplicity of names, and of opinions - To prove the justice of this statement one has only to see the extent of the nomenclature: thus some authors like Jurgensen have called it "chronic interstitial pneumonia" others, as Wilson Fox, "chronic pneumonia," Clark: Fibroid Phthisis" - Sutton and Handfield Jones:- "Fibroid degeneration" - In the earlier days Bayle and Andral described it under the name of "Melanosis."

Turning again to the opinions, of authors, we have the same want of unanimity. To give a few examples, some, such as Bayle and Andral associated this condition with tubercle in the lung - others such as Corrigan and Addison and Clark maintained just as strongly that tubercles were never present. Some again attributed the cause of this condition to prolonged chronic bronchitis - Others again, as a sequela of acute pneumonia. - But, in addition to the advantage, if it were possible, of being able to adjust these contrary expressions of opinions, there is one other reason why a simplification, and correct understanding of this subject is necessary; that is, the importance to the medical man, and the patient, of giving a definite opinion.

If these chronic indurations for instance depend upon tuberculous causes for their origin, so much the worse must it be for the prospects of life of that patient. For compared with all other chronic lung diseases, tuberculosis in whatever form, is rapid. If on the other hand a fair opinion can be given that chronic pulmonary indurations have their origin and run their course apart

from tuberculosis then the patient so affected, though he may look forward to passing the life of a comparative invalid, yet the duration of his life is much greater than in the former case. -

Having had the advantage of working as resident officer in the City of London Hospital for diseases of the chest, many opportunities have arisen for studying the varied forms of Pulmonary indurations both from a clinical and pathological aspect. The difficulties which have been enumerated above have been present to me in my study. I therefore propose in this thesis to first of all offer some critical remarks on the views already expressed by others - secondly to attempt a classification of chronic pulmonary indurations - Thirdly to give my own experience in this disease as I have met with it in practice - and lastly to arrive at conclusions which it is hoped will remove many of the difficulties which beset this subject and which will guide one's opinion in forming a diagnosis and prognosis to the right end. -

CLASSIFICATION. - A classification of chronic pulmonary indurations may be made from several standpoints - From an ætiological or pathological standpoint we may roughly divide up indurations into two classes. -

(1) Those indurations which are caused by a deposition of tubercle.

(2) Those which are the result of some simple inflammatory process in which tubercle plays no part.

Again from a consideration of the minute anatomy of the lung, we may divide chronic indurations up into groups:

(1) When the inflammation is seen to be most marked in the interstitial tissues of the lung, it may be called "Interstitial."

(2) When it is most marked in the alveoli it may be called alveolar.

(3) Lastly when the process seems to have started from the pleura, it might with justice be termed pleurogenous.

Or again from the appearance of the induration, at some time or other during its course a classifi-

cation may be made.

Thus in some advanced conditions of induration the lung is not unlike in appearance to the cirrhotic liver, and may be called cirrhosis, or from its hardness, colour, and consistency it may be termed - red - grey - or black-induration.

From the large deposition of Fibroid material at the expense of the normal lung tissue it may be called Fibroid degeneration.

Lastly from its association with ordinary Phthisis, and its similarity in appearance to Phthisis, it may be called Fibroid Phthisis.

In the most recent work on the subject of pulmonary induration, namely, the monograph by Clark, Hadley, and Chaplin[x] a classification is given at the end of the first chapter which is limited and only embraces the relations of chronic indurations to tubercle. There fibroid induration is classified -

(1) Fibroid Phthisis, or pure fibroid disease a condition in which no tubercles are to be found.

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x Fibroid diseases of the lung 1894. Griffin & Co.

(2) Fibro-Tubercular disease - a condition in which the disease was at first pure fibroid, but afterwards became secondarily infected with tubercle.

(3) Tuberculo-Fibroid disease-a condition which began as tubercle, but which afterwards took on a fibroid course, tubercle having become obsolescent.

This classification may have been made to meet the exigencies of the special nature of the book, but it is obvious that it is incomplete, as it deals with only one part of the subject, and for the purpose of this thesis will not serve us - we must therefore adopt some other form of classification.

In order to obviate the difficulties met with in classification, on account of the dissimilarity both pathological and clinical of the various forms of pulmonary induration it will be advisable not to attempt to bring all these conditions into one classified group, but to discuss them separately. - To this end we will devote our attention.

1. To those forms which have been described as "Fibroid degeneration" or Phthisis or cirrhosis.

2. To those forms which have been described as "Chronic pneumonia."

3. To those forms of chronic pulmonary induration which take place in and around tubercular depositions.

4. And lastly those which are due to some definite irritating cause such as dust.

CRITICAL COMMENTARY OF VIEWS EXPRESSED BY VARIOUS AUTHORS.

Let us before proceeding however, to discuss these four groups just enumerated, attempt to criticise the views of the more important authors who have worked in this field.

CORRIGAN. (Dub. Med.Journal 1838). This physician describes a form of induration which though it resembled tubercular Phthisis, he maintained was not due to tuberculosis.

On cutting into the lungs of his cases he fancied he saw a resemblance to the fibroid changes occurring in the liver, when affected with common or polylobular cirrhosis - He therefore gave the name of "cirrhosis of lung" to this condition.

Corrigan's view of this change, was that

fibrous tissue became largely increased in the interlobular septa of the lung - He could not bring himself to believe that the alveoli played any part in its production - Finding his lungs very diminished in size he accounted for it, by supposing that the fibroid tissue after spreading, gradually contracted, at the same time replacing the normal lung tissue. And the point he insisted upon was that the contraction of the fibroid tissue acting dynamically upon the bronchi drew the walls of those tubes apart and so caused dilations or bronchiectases.

In this view he was in antagonism to Lœnnec (Mediate auscult. 1819.) who assumed that in many cases the fibrosis was secondary to the bronchiectasis.

The main points of Corrigan's paper seem to be

(1) That this condition is not tubercular.

(2) That it is interstitial. -

It is evident that Corrigan was describing a very advanced form of induration, and that the cases which are met with exemplifying this condi-

tion are few and far between.

HANFIELD JONES. - Fibroid and allied degeneration. (Brit. For Med. Chir. Review 1854).

This author discussed the question, as to whether chronic induration of the lung or other parts of the body could be due to a diathetic condition. He formulated the theory that, when in any part of the body, as the liver, or the lungs, a fibroid condition existed, the condition was but a local manifestation of a general disease. He did not believe that inflammation was so much a starting, as an exciting cause. He also sought to group lung indurations under three headings.

1. Chronic pneumonia.
2. Hepatising pneumonia.
3. Cirrhosis.

This author introduced for the first time the question of a diathetic condition. This view has much to recommend it. It explains to a certain extent why some people should be prone to take on in some part or other of their body a fibroid condition, while others under the same conditions of life and disease altogether escape.

Many however take exception to this view, for if no other cause can be advanced, as the reason for this change taking place, except by sheltering themselves behind the word "diathesis," one's knowledge is hardly advanced beyond the invention of a phrase.

SUTTON. A prominent worker in this field brought forth corroborative evidence of the question of diathesis. He, in an exhaustive paper published in the transaction of the Medico-Chirurg. Society (1865) studied the whole question under the name "fibroid degeneration" - His conclusions were as follows:

From an anatomical point of view he held that the interlobular connective tissue was increased and the pleura thickened. The process was most commonly in the apex - He failed to notice dilatation of the bronchial tubes. -

Histologically he found a formation of fibroid tissue which invaded and destroyed the lung substance. This process was most highly developed around the bronchial tubes, and in the pleura. He did not consider the process to be due to tuberculosis. -

Clinically - the patients were well built had little wasting, and were addicted to intemperance.

He noted the absence of tubercular disease of the intestines, and found that the change in the lung was often associated with similar changes in the liver, heart and kidney - and from that, opined that a fibroid diathesis was the cause of the process. Anyone who will read through Sutton's cases can hardly fail to be strongly impressed with the view that many of them were tubercular, and that Sutton was describing really, the fibroid transformation which takes place around tuberculous deposits.

In 1868 Sir Andrew Clark published a paper in the Transactions of the Clinical Society, upon a case of chronic induration of the lungs occurring in a woman - In this paper he promulgated the view that chronic induration was a disease in which tuberculosis played no part. He more or less kept closely to Sutton's description of the disease. He insisted on the absence of tuberculosis from the fact that, clinically the base of but one lung, though advanced in disease, was affected; the other lung remaining healthy - and that when the lung came to be examined no support was forthcoming to a tubercular view. The caseous deposits found in the lung he ascribed to a retrograde metamorphosis of the fibrous tissue deposited. -

Finally Clark advanced twenty reasons why this condition should be distinguished as fibroid phthisis- tubercle having no part in its production.

Clark's paper in 1868 was the first attempt at working out for fibroid indurations of the lung a special place in medical literature - But he confined himself mainly to one point - namely - to proving that chronic indurations of the lung are not primarily due to tubercular agencies.

Wilson Fox writing in Reynolds System of Medicine, the article on chronic pneumonia in opposition to Clark and Sutton believed, that chronic pneumonia was almost always tubercular. He thought however that the exciting cause might be -

(1) Pneumonia.

(2) Bronchopneumonia.

(3) Pleurisy.

(4) Interstitial chronic inflammation.

(5) Fibroid changes in the walls of the alveoli.

He held that the chief changes in the lung from a histological point of view were to be found in the walls of the alveoli. He had no sympathy with the idea of a fibroid diathesis. Anatomically he

described the forms of chronic induration -

(1) A red induration,

(2) a black induration.

Dr. Wilson Fox was perhaps one of the strongest supporters of the tubercular view of all indurations of the lung.

BASTIAN. - Dr. Charlton Bastian wrote the Article on "Cirrhosis of the lung" in Reynolds system of medicine. In dealing with the limited side of chronic pulmonary indurations, he gave an excellent account of the disease, gathered from thirty cases. He appears to have thought that the process arose in the interstitial tissue of the lung. He did not think that the word "pneumonia" adequately represented this condition, in that there was no exudation and little or no inflammation. The cavities met with in the lungs he thought, might be formed apart from tubercle through the agency of fatty degeneration of the new tissue, gangrene, and ulceration of the tubes.

JUERGENSEN, In Ziemssen's Cyclopædia of Medicine, wrote an article entitled "Interstitial pneumonia." He looked upon chronic induration of

the lung as primarily an interstitial process, the alveoli participating secondarily. He mentions as causes, measles, broncho-pneumonia, and whooping cough as being the most common. He did not believe that tubercle is antecedent, but admits that it may be deposited secondarily.

From a clinical point of view he laid stress upon, absence of fever, slow pulse, and general good condition of the patient as distinguishing chronic induration from ordinary tubercular phthisis.

DOUGLAS POWELL gives a description of three cases of phthisis with contraction of the lungs in the transactions of the Clinical Society 1869 (Vol. vi.) These cases were evidently ones associated with tuberculosis which was probably the starting point of the affection.

WALSHE, in his text book of "diseases of the lung" described "chronic pneumonia" and "cirrhosis" as two distinct affections. Chronic pneumonia he stated to be a rare condition as a sequence to acute pneumonia but common as a concomitant of chronic tuberculosis. His description of cirr-

hosis is similar to that of Corrigan. He had often seen tuberculosis coexist with cirrhosis.

PEACOCK and GREENHOW, - in the transactions of the Pathological Society deal with the subject of pulmonary induration induced by the inhalation of irritating dust - many of these cases quoted were however cases of tuberculosis, set going by the inhalation of irritating particles.

I have hitherto said little in review of the literature of this subject concerning the work done by the French school of authors.

Lænnee, Bayle, Andral, Grisolle and Charcat, all referred in their writings to chronic pulmonary induration.

Lænnec gave a description of four cases which were fairly typical of chronic pulmonary induration. He also referred in his work (Traité de l'Auscultation Médiate) to chronic pneumonia.as following acute pneumonia. He also described the chronic induration found around tubercular deposits.

Bayle described induration under the name of "melanosis," and stated that tubercle often complicated this condition (Recherches sur la Phthisie

1810.)

Grisolle writing in (1864) ("Pneumonie") looked upon pulmonary induration as due to chronic pneumonia. He described two forms - one simple - the other arising around tubercle.

Having therefore stated in the main the views of various authors let us proceed to give an account of the various forms of chronic induration as seen at the bedside, and on the post mortem table.

As to whether "chronic pneumonia" - "fibroid phthisis," "cirrhosis," are one and the same disease seen at different stages we must leave that question until later on.

We will first of all give a description of cirrhosis, it being the typical form of chronic induration, upon which all authors are agreed.

Clinical description. Patients affected with cirrhosis may be either old or young. They give somewhat the following history - cough of a peculiar character, coming on in paroxysms, as a rule, early in the morning, on waking. - This cough is attended with the expectoration of large quantities of muco-purulent phlegm, very often

filling the porringer. In addition to this there is shortness of breath - The absence of hectic, wasting, sweating and high temperature, is most constant. The aspect of the patient is that of a person in a good state of health. The type is rather that of a well nourished man, than one affected with a wasting disease. He will frequently tell you that, were it not for the cough, shortness of breath and expectoration he would be quite well. The pulse-beats are full, measured, and slow. There is often evidence of hypertrophy of the heart. Albumen is often found in the urine. On examining the chest, it is almost always found to be a well developed one - if we omit the sinking in and retraction on the side of the disease. The retraction is most marked. By percussion - there is dulness more or less complete over the retracted side. The heart is almost always displaced towards the side of the disease. On listening with the stethoscope the sounds heard over the dull area are as a rule bronchial breathing, up to cavernous breathing - attended with large coarse mucous râles.

In addition to the heart, other organs in association with the diseased side are displaced,

thus the liver or stomach may be drawn up.

Turning now to the other side, the disease is not found there, but by careful percussion, and auscultation it can be found that the sound lung is extremely emphysematous - the lung extending right across the sternum over to the affected side. This emphysema is purely of a compensatory nature. There is hardly ever found any evidence of cirrhosis in the otherwise sound lung. This condition is of long duration going on for a number of years - perhaps forty - without any great detriment to the patient. Attacks of acute bronchitis are liable to supervene and carry the patient off. Hæmoptysis is also of somewhat frequent occurrence.

Morbid anatomy. Size of lung. On cutting into a cirrhotic lung, the pleura is found to be intensely thickened, and more or less adherent to the chest wall. From the inner surface of the pleura strands of fibrous tissue may be seen spreading into the lung between what remains of the lobules, and splitting it up into areas and plains. The naked eye appearances of the cut surface of the lung are as follows: There is hardly any lung substance proper left, the whole lung being

converted into fibrous tissue Bronchiectases are a constant accompaniment of cirrhosis. They are evidently caused by the contractile action of the fibrous tissues exerting force upon the bronchial tubes.

The liver, kidneys and spleen may also exhibit evidences of fibroid metamorphosis. The ventricles of the heart, both right and left are hypertrophied. The opposite lung is bulky and hypertrophied using the word 'hypertrophy' in contradistinction to the term 'emphysema'.-

The Ætiology - Broncho-pneumonia supervening upon measles and whooping cough, is a fruitful source of this disease. Pleurisy, prolonged chronic bronchitis, pneumonia, are also answerable for this condition. Spirit-drinking, and abuse of alcohol generally has been said to act as an excitant for this condition.-

We shall attempt to show afterwards that this condition is only the very advanced form of chronic pulmonary induration.

PATHOLOGICAL ACCOUNT - We will now give an account of the pathological anatomy of chronic induration which will serve for all types.

The Pleura is always thickened in cases of lung induration, in some instances it is enormously so, whereas in other cases the increase is only of slight extent. This thickening of the pleura is most marked over the diseased portions of the lung. The pleural cavity may be completely obliterated or the opposing surfaces may be only loosely adherent.

The Lung. - In colour the lung may be either deeply pigmented, or on the other hand, have a greyish look. The general view has been to describe chronic pulmonary induration as being deeply pigmented, but in many instances the lung, as the seat of induration, will be found to be not only, not more than ordinarily pigmented, but even of a lighter colour than the normal lung. Pigmentation, of course depends largely upon the kind of dust inhaled. Where the induration is well marked, the lightness in colour is due to the large deposition of fibroid tissue.

Size of lung. It is commonly believed that the lung, in chronic induration is much smaller

than natural, but on looking over the record of cases, and drawing evidence from my own experience, one is led to the view, that in not a few instances the lung is hardly diminished in size. Often where the disease in the lung results in contraction, the sound parts of that organ over-grow and take the place of the diseased portions.

Consistency. The consistence of the lung in diseased areas is firm, and tough not unlike leather. When one cuts into chronic pulmonary induration the knife meets with resistance, and the following appearances are noted on the cut surface. The lung seems to be intercepted in all directions by firm white lines - the greater the induration the nearer these lines are together, showing that the lung substance proper has been destroyed by the ever-growing fibroid tissue. The greatest amount of fibroid tissue is found around the small bronchi, and running in from the inner layer of the pleura - The induration in the lung may be found in various places, as a rule the seat of this lesion is in the lower two thirds, although it has been found confined more or less to the apices. Some have gone so far as to assert that the apices are the common situations for these indurations, but many cases

quoted in support of this view must be held to be tubercular. The chronic pulmonary induration may be observed in patches, circumscribed, scattered over the lung, or, as is more common, it may take on a diffused form, involving uniformly the whole or part of the lung affected.

Cavities may be found in the indurated portions. The question has been debated as to the origin of these cavities. Some authors such as Bastian and Sutton, have assumed that they result either from ulceration of the bronchi, or the degeneration and necrosis of the lung tissue, as the result of its blood supply being cut off. Others who hold the tubercular view of chronic pulmonary induration, have assumed that they are the result of tubercle.

The Bronchi are invariably thickened and dilated, the mucous-membrane lining is in a state of chronic inflammation. The dilatation may be considerable or only slight. The question as to the causation of bronchiectases need not be gone into here, beyond pointing out the contractile action of fibrous tissue may play some part in their production.

MINUTE ANATOMY. If we regard the possible situations in the lung in which chronic induration may arise it will be seen that there are three - namely:-

(1) Within the alveoli.

(2) The interstitial tissue between the lobules, and around the bronchi.

(3) The inner layer of the pleura.

In all forms of chronic pulmonary induration one or all of these three situations shows evidence of disease. It is extremely rare to find one situation alone affected, though one can often say, that the incidence of the disease has fallen upon either the alveoli or the interstitial tissue or the pleura, as the case may be - the other two situations not participating to such a great extent.

MINUTE CHANGES IN THE PLEURA. Under the microscope the inner layer of the pleura is seen to be largely composed of fibroid tissue, in the meshes of which are numerous cells, some elongated, and others dividing. In addition to this, there are seen many newly formed capillary blood vessels, this process extends into the lung, joining the interlobular tissue. It is difficult in, at all

advanced cases, to determine where the inner layer of the pleura ends, and the interlobular tissue of the lung begins.

THE INTERLOBULAR TISSUE is increased in amount. The changes seen by the microscope are described by Clark, Hadley, and Chaplin, in their monograph, as follows:

To put it briefly "The tissues present the appearance of more or less well organized fibrous tissue, containing in its meshes a few lymphoid cells - some large and thickened blood-vessels, some of which are obliterated, while the lymphatics are for the most part, so altered, as to be no longer recognisable - numerous lymphoid cells are seen in the meshes of connective tissue. - When the neighbouring alveoli, are also the seat of inflammation crowds of these cells may be seen invading the interlobular septa from them. Later these new elements become more or less perfectly organised the cells elongating and losing their nucleii. The blood vessels are seen to be increased, giving the tissue an appearance of great vascularity. In the later stages of the process the vessels have become obliterated, and the whole structure tends to become more fibroid and less

vascular and cellular."

In coming now to the changes taking place in the alveoli - several points have to be noticed.

(1) First of all many may be seen to be emphysematous. The alveolar walls can often be found to be thickened by fibro-cellular material - To quote again the above mentioned monograph, "a few cells of the epithelial lining were seen to be shed into the interior, and the alveoli were filled even in the early stages with material which was rapidly becoming organised into fibroid tissue. It differed from fibroid exudation of croupous pneumonia in that it was markedly fibroid. So again it was unlike the cellular material of catarrhal pneumonia in that there were few cells. Small capillaries may be seen perforating the walls. The further one gets into the more completely indurated lung substance, the less one sees of recognisable alveoli; the whole tissue being composed of fibroid material."

Clark, Hadley and Chaplin summarised their results as follows:- (Monograph Page 64)

(1) "Some alveoli are simply emphysematous.

(2) Some are shrunken and collapsed.

(3) Fibro cellular thickening of the walls

occurs without exudation into the interior.

(4) A sparsely cellular exudation takes place into the alveoli, which rapidly organised into fibroid material.

Other organs in the body are affected in chronic induration of the lung. -

The heart, as has been mentioned, is hypertrophied. -

The Spleen - Liver - and Kidneys, often show evidences of the formation of fibroid material.

We must now consider the ætiology of chronic pulmonary induration.

My experience has taught me that though this disease is attributed to many causes, yet only a few can be said to give rise to it with any frequency.- - But the majority of cases of chronic pulmonary induration give a history in early life of an attack of broncho-pneumonia following upon some specific fevers such as measles, or whooping cough. But although this may be the chief cause for the condition of those cases arising in early

life yet it is obvious that it offers no solution, as to the aetiology in those cases arising later in life. These cases arising in later life generally give as a cause, an attack of acute pneumonia, which failed to resolve completely - prolonged chronic bronchitis - Pleurisy - and the inhalation of irritating dust. Among the less numerous causes of the affection, one has to mention Syphilis - Bronchiectasis - and cases are on record of a traumatic origin for this affection. Alcoholism, may also be said to be responsible for it. Then there is that large group of cases which shows unmistakable evidence of chronic pulmonary induration, but in which tuberculosis plays a prominent part.

The important question arises - Can we draw any distinction between those cases, which

(1) have their origin in broncho-pneumonia -

(2) those cases which have their origin in acute pneumonia.

(3) those cases which have their origin in prolonged chronic bronchitis, and the inhalation of dust,

(4) and tuberculosis.

In the first place, cases which arise in chronic broncho-pneumonia, begin early in life, and steadily progress, for a considerable period. The characters of the disease seem to differ - thus, the whole lung seems to become affected, and from my personal observations, these cases seem to result in that extreme form of chronic pulmonary induration, which we have already described under the name of cirrhosis. - Beginning as it does at a very early age, we must not expect to find these cases lasting into the later decades - as a rule those cases which begin in this way end before the patient is forty. It must not be supposed, however, that this is the only cause by which cirrhosis of the lung is produced - all that is claimed is that the greater majority of these cases so terminate.

Those cases beginning after an attack of acute pneumonia in many instances differ essentially from the foregoing. The disease has not that steadily progressive character which is noticed in those, arising out of broncho-pneumonia. The part of the lung affected with acute pneumonia fails for some reason or other to resolve. Its diseased area becomes the seat of fibroid degeneration, but this

degeneration does not extend to such a great extent over the lung. In other words the induration remains confined and localised. The physical signs also offer marked points of distinction. In this case the percussion note is dull. The breath sounds, bronchial; but there is little or none of that coarse, moist, crepitant rale which tells us of the dilated tubes, and of a complete fibroid transformation. Patients also affected in this way do not complain, to such an extent, of great dyspnœa, and inability to perform the ordinary avocations of life.

Those cases which arise from the inhalation of irritating dust, give symptoms only when the cause has been in operation for many years. The form of induration in this case is essentially different to that met with in the two conditions enumerated above. Pathologically it rarely involves completely one lobe of the lung, but is more or less scattered in patches of induration all over. The prominent feature of these patches is their deep pigmentation.

Of those arising from chronic Bronchitis extending over a number of years, the same may be said, with regard to the circumscribed nature of

the disease - though in this case perhaps it is not uncommon to meet with a more diffuse condition of the induration at the bases of the lung.

As will be supposed in those cases where traumatic origin is present, the induration is most in evidence at the seat of the traumatism.

Those cases due to alcoholism also seem to be of a diffuse nature.

The cases of chronic pulmonary induration which arise from a tuberculous cause are altogether different to those we have enumerated above. In the first place as is evident, the common seat of tuberculosis, being the apex therefore the chronic pulmonary induration is also in that situation. - Again, in the history we find an absence of a determining cause, such as broncho-pneumonia and acute pneumonia, on the contrary there is as a precedent condition, the well defined history of tuberculosis. As the tuberculosis quiets down and dies, fibroid tissue takes its place, more particularly around the areas affected by tubercle. Hand in hand with the death of the tuberculous 'nidus' goes the gradual, and steady increase of fibroid tissue, until at last, there may be nothing left to point to tuberculous infection, beyond a few old,

smooth-walled, cured cavities and caseating centres. After this has taken place the clinical history of the case is identical with that of ordinary pulmonary induration with this exception, that an eruption of tubercle is always liable to take place.

To sum up then, the main points of distinction are these -

(1) A definite history of an attack of tuberculosis, with its characteristic symptoms and signs.

(2) Localisation of the disease to the parts of the lung most commonly affected by tuberculosis.

Perhaps the affection of other organs such as hypertrophy of the heart, fibroid transformations in the liver, spleen, and kidneys is not so often seen in these cases.

Let us now consider the main points in the symptoms of chronic pulmonary induration, taken collectively:

As to the age - chronic pulmonary induration does not affect any particular period of life, depending as it does, for its cause, upon diseases which are liable to attack at different periods.

This condition may come on from two and a half years upwards, to fifty or sixty - On account of most cases being due to an attack of broncho-pneumonia, we find, early life the period most liable to the onset of the disease.

Duration. This also varies, in some cases the disease does not last longer than five or six years - where in others it may almost extend over a life time. From the cases I have observed and from the tabulated results of Authors, probably one would not be very wide of the mark if the average duration were placed at from fifteen to twenty years. Perhaps one may say that an early onset presages a briefer duration.

Cough. This symptom is present in every case of chronic pulmonary induration. The cough has distinctive characters, it is not continuous, but comes on in paroxysms, as a rule early in the morning on awaking. After the paroxysm is over, the patient passes through a period of quiescence, until the next paroxysm comes on. The cough is attended with the expulsion of large quantities of expectoration. This expectoration is thick - often of a dark colour. It may have an intensely fœtid smell. Very often the sputum pot is nearly

filled during one of these attacks - the phlegm coming up in gushes.

Vomiting may also be brought on by the harassing nature of the cough. These characteristics of the cough and expectoration are, of course, due to that common accompaniment of chronic pulmonary induration - namely - dilatation of the Bronchi.

In a few cases however it has been noticed that this peculiar cough is absent, there being nothing pathognomonic in this symptom.

Dyspnœa - Shortness of breath is always present in pulmonary induration. Sometimes it is very marked the patient being unable to take the slightest exertion without bringing on shortness of breath.

Cyanosis - is a frequent accompaniment of chronic pulmonary induration. In these cases the extremities are almost always cold, and of a bluish tinge - this symptom goes hand in hand with the dyspnœa, and the cough.

Clubbing of the fingers - where the disease has lasted over a considerable period the fingers and toes exhibit a tendency to club.

Hæmoptysis is not an infrequent accompaniment

of pulmonary induration, but it must be remarked that it is hardly ever of that extent as is met with in ordinary phthisis - Yet, of course, when one is dealing with a case of induration arising out of tubercle, the haemoptysis may occur from the presence of that complication.

The symptoms of hectic, such as fever, sweating, and emaciation, are markedly absent in chronic pulmonary induration - Patients with extensive induration of the lungs frequently go through this disease, with a normal temperature, well covered bones, and no tendency to sweating.

Authors have pointed out, that diarrhœa is met with in this set of cases but my observations do not lead me to this conclusion.

Œdema - As the healthy lung tissue becomes replaced by useless, indurated tissue, Œdema is apt to supervene, and at the same time one may frequently detect the presence of albumen in the urine. When this is found and when it lasts for any considerable time the kidneys are generally found to be diseased.

White spots. In his first paper in 1868, Sir Andrew Clark pointed out, that patients affected with fibrosis were prone to have white

fibroid spots in the skin. I have carefully looked over many cases, but have failed altogether to note this as a common symptom.

Physical Signs. Something now remains to be said concerning the physical signs. In order to treat this part of the subject clearly, we must divide chronic pulmonary indurations into three groups. It being understood that the above symptoms enumerated are common to all forms of the disease.

The three groups are as follows:-

(1) Those cases in which the whole of the lung is affected.

(2) Those cases in which the disease is circumscribed or affects only one part.

(3) Those cases in which tubercle is closely associated.

(1) As to the first condition:

On inspection, the side diseased is contracted and misshapen, it moves scarcely at all on respiration, but although this is the general appearance, yet the chest is generally well covered, the shoulder droops on the affected side, and frequently a slight curvature of the spine may be noted. If the disease be left-sided, the heart's apex beat is

seen to be displaced into or towards the axilla. If it be right-sided disease, the apex beat of the heart may be seen and felt beating in any position between the normal, and the right nipple line, or even further. -

On percussing the diseased side of the chest the note is impaired up to dulness over the more solid and condensed portions of the lung. The liver and stomach note may also be detected higher up than is normal - absolute dulness is generally met with at the base of the lung.

Turning to the non affected side the note here is found to be hyperesonant. This hyperesonance extending over the sternum to a few fingers'breadths.

Vocal fremitus as a rule is increased over the affected areas, but sometimes, owing no doubt, to the thickening of the pleura it may be found to be absent.

Auscultation. The breath sounds are exaggerated, and in the place of normal sounds may be heard bronchial, or cavernous breathing, attended with coarse mucous rales. On testing the voice sounds they are broncho phonic, or even pectoriloquous. Over the sound side the breathing is simply exaggerated, attended with occasional rhonchi, and

give crepitations at the base of the lung.

(2) As to the second division.

In this class of cases the physical signs are not so pronounced, and in some respects differ from those just enumerated.

In a lung in which induration is localised; or in circumscribed patches the retraction of the chest is little or not at all marked. The percussion note is only impaired over the affected areas. The intervening lung tissue gives a hyperesonant note. The heart too is not displaced to any extent - On auscultating such a lung, at the seat of the disease, one may hear perhaps bronchial breathing attended with crepitations, but more frequently over the diseased area especially if the pleura be thickened one can hear nothing, or at most very weak breath sounds - there is an absence of those loud, coarse, mucous rales, and very rarely does one get Bronchial breathing or bronchophony.

(3) On looking at a chest belonging to the third variety, the first thing one notices is that the chest is not well covered. The patient has not that fine physique observed in the other conditions.

There is immobility, and retraction at the apex of the lung. The note here is impaired or dull. On listening over this area one can hear bronchial breathing, attended with crepitations which are of a finer character than those heard in the other forms of the disease. Usually there is evidence of excavation. The apex beat of the heart may be drawn upwards if the disease is on the left side, but little if at all altered in position if the disease is on the right side. The lower two thirds of the lung presents to the ear and fingers normal signs. The opposite lung instead of being simply emphysematous generally shows evidence of disease more or less advanced at the apex, indeed the condition here described, is simply one of old cured tuberculosis.

Diagnosis.

In these several conditions a diagnosis has to be made between them, and various other lung diseases. Pneumonia, when resolving, sometimes presents similar signs, but in this case, a history of the onset of the disease, showing a short duration should prevent a mistake being made. For the same reasons, no difficulty should be experi-

enced in diagnosing between chronic pulmonary induration and anurysms or morbid growths. For in addition to this, contraction of the chest is absent in morbid growths, and prëssure symptoms are present in the latter conditions.

Thickened pleura as a result of old empyemata present more difficulty in diagnosis, for frequently lungs present signs of induration where the thickened pleura has been long in evidence. But in cases of simple pleural thickening the chest is never so contracted, and there is scarcely any cough, attended with that amount of expectoration met with in chronic induration. In thickened pleura also, the voice sounds, and breath sounds are almost absent. But this it must be remembered is not an altogether unknown accompaniment in chronic induration.

A few typical cases, of the four main groups of chronic induration, may be here briefly enumerated.

I. As representing the interstitial form. A female - single - well developed - with dark hair and eyes - aged 28, was admitted to the wards of the "Victoria Park Hospital, for diseases of the

chest," in 1892.

State on admission. - Patient looks well. She complains of great weakness, pain over left side and back. Cough very troublesome, especially in the morning when she has paroxysms of coughing accompanied with profuse expectoration of offensive phlegm - paroxysm of coughing sometimes followed by vomiting, dry skin - temperature normal - fingers and toes clubbed - pulse slow - regular - 65.

Family History. - good - no history of phthisis or Rheumatism - father has winter cough but is alive and well.

Personal history. Had measles followed by whooping cough, at the age of four - Has had a cough ever since both in summer and winter time. Has always suffered from dyspnœa on exertion - Had had no other illness.

Physical signs, on inspecting chest, flattening, contraction, and diminution of movement all over left side. Apex beat seen and felt in 5th. interstitial space in the post axillary line.

Percussion note dull all over left side from apex to base. Right side note hyperesonant.

Auscultation. Heart sounds, normal regular. Left lung, breath sounds bronchial, and pectorilo-

quous, accompanied by coarse gurgling crepitations. Right side emphysematous.

Urine - slight cloud of albumen, otherwise normal.

Patient discharged, and was not re-admitted during my term of office.

II. The pneumonic Type. -

Male - 52 years - well grown, - occupation bookmaker's clerk.

Family History, none of tubercle or rheumatism.

Personal history - pneumonia when aged 38, never quite well since - no other illness.

State on admission - patient looks ill - has been spitting up mucous streaked with blood - marked dyspnœa - lips and finger nails of a bluish tinge, especially so after exertion.

Physical signs - inspection contraction - loss of movement - diminished expansion, all over right side especially at base. Heart seen and felt in epigastrium.

Percussion - Right base dull.

Auscultation - breath sounds almost absent, a few scattered crepitations and friction sounds over Right base behind - Right apex and left lung

normal - No Tubercle bacilli found in sputum - Heart sounds, normal.

Urine - trace of albumen.-

ON P. M. EXAMINATION.

Heart displaced to right otherwise normal.

Right lung, pleura adherent all over, thickened at base, lung small shrunken, the lower lobe feels hard and tough, is greyish when cut into, the bronchi are dilated with thickened walls, which are inseparable from the general mass of fibrous tissue, composing the lower lobe, and which almost entirely replaces the lung tissue, no evidence of tubercle. Right apex and left lung normal except at the edges which show emphysema. Other organs healthy. -

III. Type of case due to prolonged chronic Bronchitis.

Male, aged 65. Occupation dock-labourer.

Family history - no history of phthisis, mother died of "dropsy."

Personal history - has had bronchitis for twenty years - no other illness.

State on admission - Patient emaciated has troublesome cough - marked dyspnœa and pro-

fuse white frothy expectoration.

Physical signs inspection, contraction and impaired movement all over right side of chest - heart apex beat felt and seen in epigastrium.

Percussion note dull from apex to base of right side, left lung normal.

Auscultation over right side breathing bronchial attended with coarse crepitations - left side gives evidence of emphysema.

Temperature normal.

Urine normal, no albumen.

Pulse slow, regular.

IV. Type of tuberculous case.

Man - aged 23 - occupation shop assistant.

Family history - mother died of phthisis, no other member of family with phthisis.

Personal history. Well and healthy until six years ago, when he began to cough, and lose flesh, had night sweats, and hæmoptysis.

State on admission - marked emaciation, weak - troublesome cough - Expectoration blood-stained free - clubbing of fingers - pulse 100. Temperature 99-101.

Physical signs on inspection, the chest showed

marked flattening, over upper half of left side - with loss of movement over whole of left side and right apex. Apex beat in 5th. interspace in left nipple line.

On Percussion - note dull all over left side and impaired at right apex.

Auscultation. Breath sounds bronchial, with coarse crepitations all over left side, and at right apex. Vocal resonance and fremitus increased over these areas.

Tubercle bacilli were found in sputum.

Pulse rate and temperature were raised.

Night sweating marked.

Urine normal no albumen.

Patient had a severe attack of hæmopysis, and died after 3 weeks of the usual symptoms of tubercular disease.

Post Mortem examination.

Heart slightly displaced to left side.

Pericardium adherent to left pleura.

Heart muscle small flabby, thin walled and pale in colour - no valvular disease.

Left lung. Pleura thickened and adherent all over, thickening marked at apex.

Lung small, tough to the knife, several old, small cavities, and dilated bronchi, especially at apex, surrounded with bands of fibrous tissue, greyish in color, and radiating all through lung substance. At left base there is evidence of recent advance of tubercle.

Right lung apex showed active tubercular advance, with cavitation.

SUMMARY AND CONCLUSION.

We have now discussed at some length the various forms of chronic pulmonary induration.

We have shown that these forms differ both clinically and pathologically. It now remains to summarise our results and state the conclusions to which we have come.

Chronic Pulmonary indurations may then be divided into four main groups:-

(1) The simple or interstitial form.

(2) The pneumonic.

(3) The tubercular form.

(4) The chronic induration, as the result of inhalation of irritating dust, and pressure on the lung by other structures.

The simple or interstitial form begins generally after an attack of broncho-pneumonia, following measles or whooping cough, it invades progressively the whole of the lung affected. A constant accompaniment of this affection is dilatation of the bronchi. Histologically the increase of fibrous tissue is most plentiful in the interstitial tissue between the lobules. Organisation of fibrous tissue in the alveoli though often present, is not nearly so marked.

The disease begins as a rule in the lower two thirds of the lung, and spreads upwards. On looking at the cut section of the lung, one sees the lung tissue supplanted by this over-growth of fibrous tissue - and in very advanced cases, the whole lung is shrunken, sometimes to the size of the fist, and presenting a condition in which there is simply fibroid tissue and dilated bronchi. Cavities, owing to the cutting off of the blood supply, and consequent necrosis of tissue, may often be observed. The advanced form of this group of pulmonary induration is known under the name of Cirrhosis.- The disease lasts over a considerable number of years, perhaps twenty. Another cause of this condition may be prolonged chronic bronchitis.

Although it may begin, occasionally, late in life; yet as a rule owing to broncho-pneumonia being the predominating cause, it is principally a disease beginning in early life.

Clinically the disease has well marked features. The symptoms are those of cough coming on in paroxysms, attended with the expulsion of large quantities of phlegm and dyspnoea. There is an absence of sweating. The temperature is not raised.

The general appearance of the patient is robust, and but for those symptoms noted above, they would be in the possession of fair health.

Amongst other signs, we must notice, clubbing of the fingers. Albuminuria, Hypertrophy, and displacement to the affected side, of the heart. The pulsations of the heart being slow, full, measured, and infrequent.

Turning to the lung, there is contraction and immobility of the affected side. Dulness on percussion. Bronchial breathing attended with coarse mucous rales. The other lung is emphysematous. The kidneys, spleen, and liver, may all be enlarged, and the seat of fibroid changes. In fact looking at this form of chronic induration, it is not too much to say that it has definite signs, and symptoms,

course and duration, and pathological appearance, which enable us to distinguish it from other forms of chronic induration.

We have seen in the earlier parts of this thesis that some authors believe in a diathetic condition as playing a part in the production of chronic indurations. One would not be far wrong in attributing a fibroid diathesis as in some part answerable for this form of induration, bearing in mind the tendency of the disease to attack not only the lung, but other organs in the body.

II. The pneumonic form differs from the others. It begins as a rule after acute pneumonia, or that form of pneumonia known as pleurogenous. In not a few instances, pneumonia especially, if of an ill-defined type, fails to resolve, in other words chronic pneumonia may supervene on the acute. Here the alveoli of the lung are principally affected. It must not be supposed that the interstitial tissue escapes, but what is contended is that the interstitial tissue is not nearly so much implicated, as the alveoli themselves. This form tends to begin later in life. Owing to the interstitial tissue partially escaping the fibroid

transformation, there is not such a tendency to dilatation of the bronchi. When the lung is cut into, the affected part has a more pneumonic appearance, the organised fibroid tissue being not so much in evidence. The disease begins, almost uniformly, at the base of the lung. It is not of that slowly progressive, and invasive character, as is the interstitial form. It remains localised to the part first attacked. It probably lasts as long as the first form, but the symptoms and signs are essentially different.

Here there is no definite sequence of symptoms which enable one to label this, as a definite disease. The cough, and expectoration and shortness of breath have not that definite character. When one examines the chest one finds at the base a dull patch, the rest of the lung may be healthy. On listening over this patch the breath sounds are weak, perhaps bronchial attended with a few crepitations. Displacement of the heart is not common. Fibroid disease of the other organs, and Albuminuria are hardly ever present. Patients affected with this pneumonic form do not suffer nearly the incapacity and inconvenience which is present in the first form, in fact, it is a milder

disease.

III. Pulmonary induration complicated with tubercle, is situated at the common seat of tubercle - namely - the apex. The lung here is thickly interspersed with fibroid tissue, around old caseous nodules and cavities. The caseous masses when examined by appropriate means, show the presence of tubercle bacilli. This disease differs from the others in the following particulars:

(1) The onset is different. There is no definite illness, or cause. It begins insidiously.

(2) For a long time the disease presents all the features of ordinary tuberculous phthisis, such as, wasting, sweating, haemoptysis, high temperature.

(3) Tubercle bacilli can almost always be found.

(4) The aspect of the patient is that of one affected with tuberculosis.

(5) The chest is narrow, the sternum depressed. The chest has never that well formed appearance as in other forms.

The disease is situated at the apex. The

bases more or less escaping. The opposite lung often presents signs of tuberculosis at the apex. The heart is not as a rule hypertrophied. Displacement is not so noticeable. The absence of that slow, measured, infrequent beat, can almost always be noted in these cases. Albumen in the urine is not often present. The duration of the disease is much shorter than that of the other forms.

IV. The fourth group, namely, chronic induration arising from the inhalation of irritating particles, and that arising from pressure, produced by morbid conditions of other organs in the chest, need not long detain us.

The effect of the inhalation of irritating dust upon the lung was studied carefully by Peacock and Greenhow. So far their observations go to show that these irritating dusts set up in various parts of the lung chronic pneumonic inflammation. Greenhow noticed, in connection with this disease, a cool skin, quiet pulse and an asthmatic cough, to distinguish it from tubercular phthisis. This form of induration was studied among men, who had followed the occupation of coal miners, and the

lungs when cut into, were found to have in addition to the patches of chronic induration, a deeply black colour. The lung on pressure exuding an inky juice, which stained the fingers.

On reading over these cases however, one cannot but come to the conclusion, that many were instances of tubercle in which, choice of occupation had imprinted special characteristics.

The lung may also become indurated in parts, through the agency of pressure. Thus, when an anurysm has pressed on the lung, at the seat of pressure chronic interstitial inflammation, together with chronic pneumonic inflammation has been set up. So also glands in the neck and mediastinum pressing on the apices or edges of the lung, have caused the same result. New growths of various kinds are sometimes answerable for this condition.

Dr. Andrew of St. Bartholomew's Hospital maintained that a hypertrophied heart was capable of causing induration of the lung, by pressure.

We have seen therefore that although chronic induration, when divided up into groups, such as we have done above, presents many different clinical and pathological aspects. - Yet, that

the same process, namely, the production of fibroid tissue, at the expense of the lung tissue is at work. In some forms we have shown that over-growth of fibrous tissue in the interstitial tissue plays the chief part. In others, that the alveoli play the chief part. In others too, tubercle plays the chief part. Because these various forms differ in their clinical history, it has been necessary, in order to recognise them, to divide them into groups. We venture to think that if these four groups are kept clearly in mind, the disease of chronic induration of the lung, will be more easily recognisable; and much of the confusion, which has hitherto arisen, will be avoided.